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## Prevention

### PERSONAL BLACK CARBON LEVELS INFLUENCE AUTONOMIC FUNCTION IN METABOLIC SYNDROME: INSIGHTS FROM THE PROSPECTIVE BEIJING AIRCMD STUDY (NIH FUND)

Poster Contributions

Poster Sessions, Expo North

Monday, March 11, 2013, 9:45 a.m.-10:30 a.m.

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**Background:** Environmental factors such as air-pollution has been postulated to synergize with traditional risk factors in determining cardiovascular risk. We assessed the relationship of particulate matter exposure [personal black carbon (BC) exposure and PM<sub>2.5</sub>] with autonomic function and functional measures of vascular risk in an urban megacity.

**Methods:** 65 adult non-smokers with metabolic syndrome underwent continuous assessment of personal level BC using microaetholometers during a 5-day period. Regional PM<sub>2.5</sub> were collected at 3 urban sites and averaged. Autonomic function, arterial compliance and digital arterial endothelial function was measured in the final 24 hours. HRV measures included both frequency and time domain measures. The relationship between various BC and PM<sub>2.5</sub> lag-structures (1/3/5 day lags and moving averages) and outcomes were assessed.

**Results:** Mean personal BC was 4.78 µg/m<sup>3</sup> while mean ambient PM<sub>2.5</sub> was 63.3 µg/m<sup>3</sup>. An increase of 1µg/m<sup>3</sup> in BC 1-day prior was associated with an increase of 24-h heart rate of 42.2% [95% CI, 3.2% to 81.2%]. On adjustment of covariates, 1-day lag BC was negatively associated with HF power, rMSSD, and R-R interval. Ambient PM<sub>2.5</sub> exposure showed a different pattern with 5-day and 3-day moving averages being significantly associated with lower HF and TP on multivariate analysis. There was no relationship between arterial PWV, augmentation index and digital endothelial function and any of the exposure measures.

**Conclusions:** Changes in BC levels and PM<sub>2.5</sub> are associated with alterations in autonomic function, albeit with different temporal relationships suggesting that compositional differences may differentially impact autonomic function. Our findings have implications for the association between anthropogenic sources of air-pollution and cardiovascular events.